



Baroreceptor reflex during forced expiratory maneuvers in individuals with chronic spinal cord injury



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ABSTRACT

Pulmonary and cardiovascular dysfunctions are leading causes of morbidity and mortality in patients with chronic Spinal Cord Injury (SCI). Impaired respiratory motor function and decreased Baroreflex Sensitivity (BS) are predictors for the development of cardiopulmonary disease. This observational case-controlled clinical study was undertaken to investigate if respiratory motor control deficits in individuals with SCI affect their ability to perform the Valsalva maneuver, and to determine if a sustained Maximum Expiratory Pressure (MEP) effort can serve as an acceptable maneuver for determination of the BS in the event that the Valsalva maneuver cannot be performed. The BS outcomes (ms/mmHg) were obtained using continuous beat-to-beat arterial blood pressure (BP) and heart rate (HR) recordings during Valsalva or MEP maneuvers in thirty nine individuals with chronic C₃–T₁₂ SCI. Twenty one participants (54%) reported signs of intolerance during the Valsalva maneuver and only 15 individuals (39%) were able to complete this task. Cervical level of injury was a significant risk factor ($p = 0.001$) for failing to complete the Valsalva maneuver, and motor-complete injury was a significant risk factor for symptoms of intolerance ($p = 0.04$). Twenty eight participants (72%) were able to perform the MEP maneuver; the other 11 participants failed to exceed the standard airway pressure threshold of 27 cm H₂O. Neither level nor completeness of injury were significant risk factors for failure of MEP maneuver. When the required airway pressure was sustained, there were no significant differences between BS outcomes obtained during Valsalva and MEP maneuvers. The results of this study indicate that individuals with high-level and motor-complete SCI are at increased risk of not completing the Valsalva maneuver and that baroreflex-mediated responses can be evaluated by using sustained MEP maneuver when the Valsalva maneuver cannot be performed.

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1. Introduction

In the United States, among the 275,000 people who are living with chronic SCI (Devivo, 2012; NSCISC, 2014), cardiovascular and pulmonary dysfunctions are leading causes of morbidity and mortality (Eigenbrodt et al., 2000; Frankel et al., 1998; Garshick et al., 2005; Walter et al., 2002). After SCI, impairment to descending sympathetic pathways causes decreased baroreflex sensitivity (BS) associated with poor baroreflex-mediated cardiovascular responses and deterioration in the inability to control heart rate (HR) in response to blood pressure (BP) changes (Grimm et al., 1998; Phillips et al., 2012; Wecht et al., 2003). This is pertinent in a

population predisposed to cardiovascular disease, as analysis of BS is predictive of future cardiovascular events (Koutelou et al., 2009; La Rovere et al., 1998): a blunted baroreceptor reflex can increase the risk of myocardial infarction and stroke due to poorly regulated BP (La Rovere et al., 2008). Accurate assessment of cardiovascular function via baroreceptor control is therefore an important tool in the management of cardiovascular dysfunction secondary to SCI.

Several non-invasive methods have been developed for evaluating BS, including the analysis of reflex responses to pharmacological or mechanical manipulations of baroreceptors, or analysis of spontaneously occurring changes in BP and HR (Osterhues et al., 2000). A standard approach that can be used to measure spontaneous BS without major redistribution of blood volumes is the Valsalva maneuver (Gorlin et al., 1957; Grimm et al., 1998; Novak, 2011; Porth et al., 1984), a forced expiration with a closed mouth and nose, during which an airway pressure of at least 27 cm H₂O (20 mmHg) is sustained for 15–20 s (Benarroch et al., 1991; Porth et al., 1984;

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Table 1
Participant Demographics and Performance of Valsalva and MEP maneuvers.

Subjects (n = 39)		Age (years)	Sex	Height (in)	Weight (lbs)	Level of SCI	AIS category	Time after SCI (mo)	Completion		Signs of intolerance of Valsalva	
									Valsalva	MEP		
Cervical (n = 27)	Complete (n = 16)	A58	40	M	70	230	C3	A	21	N	N	P
		B18	56	M	72	155	C3	B	29	N	Y	AD
		A54	32	F	68	125	C4	A	114	N	Y	
		A64	49	M	75	155	C4	A	405	N	N	AD
		A65	29	M	68	180	C4	A	10	N	Y	
		B06	42	F	67	123	C4	B	70	N	N	
		B16	60	M	71	220	C4	B	31	N	Y	
		B21	30	M	73	165	C4	B	64	N	Y	
		B22	51	M	70	180	C4	B	37	N	Y	P
		A67	30	F	65	128	C5	A	48	N	N	P
		A71	25	F	68	195	C5	A	85	Y	Y	AD
	B11	25	M	70	185	C5	B	119	N	N	P	
	B19	40	M	74	177	C6	B	6	N	Y	S	
	A68	34	M	70	125	C7	A	33	Y	Y	P	
	B24	21	M	67	125	C7	B	25	N	N	P	
	A69	28	M	72	165	C8	A	44	N	Y	P	
	Incomplete (n = 11)	C33	59	M	69	145	C2	C	6	N	Y	
		D35	60	M	71	220	C2	D	35	Y	Y	
		D38	42	M	70	245	C2	D	155	Y	Y	
		C27	58	M	70	190	C4	C	47	N	N	
		C30	19	F	67	94	C4	C	12	N	N	P
		C31	24	F	66	108	C4	C	27	N	N	AR
		C34	20	M	76	140	C4	C	53	N	N	P
		C38	18	M	72	130	C5	D	420	N	Y	P
		D42	72	M	71	230	C5	D	33	Y	Y	P, AR
		C26	33	M	72	165	C6	C	4	N	Y	S
	D40	33	M	31	135	C7	D	39	Y	Y		
Thoracic (n = 12)	Complete (n = 7)	B20	28	M	65	128	T2	B	53	N	Y	S
		A57	27	F	73	165	T4	A	45	N	N	P
		A59	26	M	73	140	T4	A	27	Y	Y	AD
		A70	32	M	74	210	T4	A	50	Y	Y	
		B25	43	F	61	125	T4	B	52	N	Y	P, AR
	Incomplete (n = 5)	A66	48	M	72	170	T6	A	82	Y	Y	
		A55	35	M	68	165	T11	A	50	Y	Y	
		C32	35	M	76	201	T2	C	67	Y	Y	
		C28	29	M	70	160	T5	C	56	Y	Y	
		D41	44	F	65	180	T8	D	40	Y	Y	
Mean ± SD		38 ± 13	N/A	69 ± 7	163 ± 37	N/A	N/A	67 ± 88	N/A	N/A	N/A	

Signs of intolerance of Valsalva maneuver: presyncope (P), syncope (S), arrhythmias (AR), and/or bouts of autonomic dysreflexia (AD).

Walker and Cutting, 2010). The increase in intrathoracic pressure decreases venous return and cardiac output which results in cardiovagal tone withdrawal during initial 4–7 s (early phase II), followed by an increased sympathetic vasomotor activity during consecutive 13–16 s (late phase II), and increased parasympathetic cardiac activity upon airway pressure release (phase IV) (Daroff and Aminoff, 2014; Kihara et al., 1998; Novak, 2011; Persson and Kirchheim, 1991; Sandroni et al., 1991). This maneuver is repeatable, non-invasive, and easily administered bedside and therefore is a common clinical test used in both the non-injured and SCI populations (Airaksinen et al., 1993; Grimm et al., 1998; Phillips et al., 2012; Previnaire et al., 2012; Rostagno et al., 1999; Vogel et al., 2005). Adequate performance of the Valsalva maneuver requires considerable expiratory effort associated with recruitment of expiratory muscles. In SCI, however, the ability to recruit these muscles for forced expiration is impaired and depends on the level of injury, with cervical and upper thoracic levels experiencing significantly diminished airway pressure generation as a result (Ovechkin et al., 2010; Schilero et al., 2009). It would logically follow that patients with cervical and high-thoracic injuries would have trouble performing the Valsalva maneuver. However, there are no reports in the literature to indicate whether SCI would render the Valsalva maneuver difficult (Grimm et al., 1998; Johnson et al., 1969; Previnaire et al., 2012). Our previous work has shown that the

majority of individuals with chronic SCI are able to generate substantial and sustained airway pressure during the MEP maneuver (Aslan et al., 2013; Ovechkin et al., 2010).

The purpose of this study was thus to investigate if individuals with SCI are able to perform the Valsalva maneuver, and to determine if the MEP maneuver can serve as an acceptable replacement for determination of BS in the event that the Valsalva maneuver could not be performed. To be consistent with the clinical standards for the Valsalva maneuver, the MEP effort required participants to sustain an expiratory airway pressure above the standard threshold level of 27 cm H₂O, equal in duration to early phase II of the Valsalva maneuver (Kihara et al., 1998; Novak, 2011; Persson and Kirchheim, 1991; Sandroni et al., 1991). We hypothesized that the BS responses to the MEP maneuver during phases II and IV are representative of those observed during early phase II and phase IV of Valsalva maneuver.

2. Methods

2.1. Demographic and clinical characteristics

Research participants were recruited from the outpatient pool at Frazier Rehabilitation and Neuroscience Institute in Louisville, KY, or were referred to the study by a clinician. After approval by the University of Louisville Institutional Review Board, partici-

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